ON THE

DIAGNOSIS AND TREATMENT

OF

APOPLEXY.

BY

THOMAS STRETCH DOWSE, M.D, F.R.C.P. Ed.,

PHYSICIAN TO THE HOSPITAL FOR EPILEPSY AND PARALYSIS,

REGENT'S PARK.

A Lecture

DELIVERED AT THE HOSPITAL, MAY 2ND, 1881.

LONDON:

T. DANKS, STEAM PRINTING WORKS,

16 GREAT WINDMILL STREET, HAYMARKET, W.

1881.

This Lecture has been printed by request.

THOMAS STRETCH DOWSE

14 Welbeck Street,
June, 1881.

ON
THE DIAGNOSIS AND TREATMENT
OF
APOPLEXY.

Gentlemen,—I have chosen for the subject of my lecture of to-day "The Diagnosis and Treatment of Apoplexy." At first sight the subject may appear to possess less interest than many other subjects in connection with diseases of the brain and nervous system; yet I cannot fail to be impressed with the fact that the practitioner of medicine is frequently summoned to attend such cases, where the question of diagnosis is not always an easy matter, and where the question of prognosis becomes doubtful; and I venture to say that the question of treatment is not unfrequently more difficult than either. We will, however, take it for granted that in no case of apoplexy can the appropriate remedies be applied unless we are able to diagnose with a fair amount of accuracy not only where the lesion of the brain is situated, but it is also necessary for us to comprehend the nature as well as the extent of the lesion which has given rise to the fit of apoplexy. I hope in the remarks which I am about to make I shall show you in a clear and practical manner that my assertions are not groundless.

I need scarcely remind you that an apoplectic state of the brain may be brought about by a disordered condition of the blood. By a diseased condition of the circulatory apparatus for the transmission of the blood either in the heart itself or, as is more frequently the case in the

vessels of the brain. Apoplexy may give rise to death in a few hours or in a few days, or it may result in paralysis. Yet, on the other hand, the concussion and compression to which the brain has been subjected may pass off and leave the patient free from any objective signs which may lead us to conclude that some serious injury to the brain has been inflicted. It would be unwise, in my opinion for all practical purposes, to restrict the term "apoplexy" to those cases only which are associated with sudden hæmorrhage into and ploughing up the substance of the brain, and it would be in a measure unwise to apply the term apoplexy to those cases of coma which are due to renal and vascular changes, and which are invariably associated with a chronic inflammation of the pia mater and arachnoid membrane, giving rise to a sodden state of the brain due to the extravasation of serous fluid from the blood. I am inclined to adopt Dunglison's definition of the term apoplexy "as a form of disease characterised by the sudden diminution or loss of conscious sensation and voluntary motion usually caused by pressure on the brain." If we apply the term apoplexy to those cases only where by sudden hæmorrhage into the brain without premonitory symptoms we have complete and immediate loss of animal life, then I must say that such cases are extremely rare.

I must briefly in the first place call your attention to the circulation of the blood in the brain. We have two systems of vessels which supply the brain with blood, namely, the basal system and the cortical system. To the elaboration of these systems we are indebted to M. Heubner, M. Duret, and M. Charcot. The basal system comprises the circle of Willis and the trunks of the cerebral arteries in connection with this circle. If the base of the brain be exposed and the arteries gently raised with the handle of a scalpel the numerous offshoots can be readily traced as they pass to supply the various ganglia. The grey matter is much more vascular than the white. We know that the post-cerebral arteries supply the sphenoidal and occipital lobes, the anterior arteries supply the anterior lobes and a large extent of the interior surface of the hemispheres as well as the subjacent regions of white matter, while the middle cerebral or sylvian arteries distribute themselves over the inferior frontal and ascending convolutions, the parietal lobe, and dip into the subjacent parts of the medullary

centres, and, moreover, furnish branches to the optic thalami and corpora striata. The great psycho-motor centres of the brain are thus supplied by the branches of the sylvian or middle cerebral artery, and we shall find that this artery and its branches are more frequently the seats of hæmorrhage, thrombosis, and emboli than are the other arteries of the brain ; in fact, the anterior cerebral and the posterior cerebral arteries are by comparison rarely the seats of thrombosis, embolism, or hæmorrhage. It is only reasonable to come to the conclusion that we should, as a matter of course, find that the more common seats of hæmorrhage are in the arterioles which spring almost immediately from the great arterial trunks which have been just referred to, for they resemble, as Heubner describes them, the straight roots at the base of a forest tree, and more than this, they are not anastomosing arteries for they terminate in pencil-like tufts of capillaries ; and again, the road from the heart and main arterial trunks to these vessels is short and more direct than in those vessels which supply the grey matter of the convolutions of the brain and which anastomose in every direction. Hence we find rupture of the basal system of vessels much more frequent than rupture of those vessels which form the cortical system. Pathology, indeed, teaches us the truth of this, for, as a matter of fact, in all large hæmorrhages, and especially those which fill the ventricles with blood, we shall find by careful dissection that rupture has occurred either in the lenticulo-striate or the lenticulo-optic arteries, which are direct branches of the sylvian artery, or we shall find that rupture has taken place in those branches which are sent to the optic thalamus by the posterior cerebral artery, namely, the posterior internal optic artery and the posterior external optic artery ; and just in the same manner and for the same reason that these arterioles are the most liable to rupture, so they are most frequently the seats of miliary aneurisms.

According to Andral and Durand-Fardel the vessels which I have just referred to, and which supply the corpus striatum and optic thalamus, were the seats of hæmorrhage in 162 out of 199 cases. Gintrac, whose laborious statistics in cerebral hæmorrhage are worthy of note, states that in a total of 751 cases of all ages he found that it occurred in different situations of the brain, in the following ratio :—Meninges 172, cortical substance of the

brain 45, middle lobes of the brain 127, posterior lobes 33, anterior lobes 17, corpora striata 72, optic thalami 38, pons and cerebral peduncles 76, medulla 2, cerebellum 55. From these statistics, and from common experience, no less than from the vascular anatomy of the brain, it is evident that the middle lobes of the brain in the neighbourhood of the corpus striatum and optic thalamus are the most frequent seats of hæmorrhage, and thus the most frequent sources of apoplexy.

I should like to draw your attention once more to the researches of Gintrac, in reference to the age when hæmorrhages are most liable to take place. He states that out of 658 cases he found—

Birth	to	10	years	...	...	15	cases.
11 years	to	20	,,	...	...	24	,,
21 ,,	to	30	,,	...	...	44	,,
31 ,,	to	40	,,	...	...	74	,,
41 ,,	to	50	,,	...	...	98	,,
51 ,,	to	60	,,	...	...	129	,,
61 ,,	to	70	,,	...	...	152	,,
71 ,,	to	80	,,	...	...	110	,,
81 ,,	to	90	,,	...	...	12	,,
						658	

Now, from these statistics alone, in reference to age and localisation, we gather very important knowledge and information, which will help us to a differential diagnosis in regard to the seat, or what may be the probable seat, of lesion when we are called to visit a person who is in an apoplectic fit; and we must never forget that the same symptoms which are produced by hæmorrhage may be produced by cutting off the blood-supply by thrombosis or by embolism. My own experience leads me to the conclusion that thrombosis of the cerebral vessels, and more particularly of the cerebral veins, is not unfrequently a cause of apoplexy in cases of septic blood poisoning, where the initial symptoms of the illness have been referable to a state of so-called sub-acute rheumatism, but where, in fact, the blood has become disorganised by septic agency, and defibrination has taken place in the venous blood current, producing such defect in the cerebral circulation, ending in dissolution and death.

Although, as we have seen, apoplexy may occur at any

age, there can be no question that the liability to these attacks increases from 20 years and upwards, and in, perhaps, the majority of cases the age is over 60. This is said by many observers to be due to degeneration in the coats of the vessels, either from gouty or atheromatous change, or from changes due to Bright's disease; or it may be from syphilis; and I think it is highly probable that where apoplexy takes place between the age of 35 and 45 years, that syphilitic change of the vessels is by no means an uncommon factor.

Before entering upon the consideration of the regional diagnosis of certain brain lesions which give rise to apoplexy, I would draw your attention to a condition of the brain which produces a state resembling it in many respects, but which differs from it in many and very important particulars. I allude to what is referred to by Trousseau as "apoplectiform cerebral congestion," but which is, according to this learned authority (and I may say in passing that the views expressed by Dr. Moxon recently at the College of Physicians tend to the same direction), due rather to a bloodless state of the brain, and is allied to epilepsy and eclampsia. By way of an example of this condition, a man with or without premonitory symptoms falls down suddenly in a state of apoplexy, in which he may remain for some hours; but the application of cold to the face, and sinapisms to the calves, or an injection of common salt by the rectum, soon restores him to a state of consciousness; and on the following day all the symptoms have disappeared.

Trousseau * gives many cases of this kind, and I would ask you to allow me to narrate one of them :— "Some time ago I was fetched in haste to see a neighbour, aged 70, who had been seized with apoplexy on the Boulevards. He had been insensible for a quarter-of-an-hour, but was recovering his senses as I arrived. He did not yet recognise me, however, and looked vacantly round, moving his arms and legs about without being conscious of it. His lips and nose were swollen, and his eyes injected. By degrees, and within a few hours, he recovered entirely, without my having had recourse to any active measures. His valet then informed me that his master had, in the last two or three years, had several attacks of the same kind, and that the symptoms had passed off in

* Trousseau, "Clinical Medicine," vol. i.

the same way, once after bleeding, and on the other occasions after a mustard foot bath. In the same year I was consulted by a solicitor from the country, aged 35, who in the course of the previous six months had suffered from three apoplectic fits. He had been bled and purged on each occasion to his great satisfaction, and leeches were applied once a month round his arms. The last attack had occurred as he was going upstairs, on his return from some important pleadings. His head had struck against the stairs. The apoplectiform phenomena had lasted an hour at the most; and when I saw him his intellect, sensibility, and power of motion were perfectly normal. I can with difficulty believe that apoplexy occurs in persons aged 37, particularly when the attacks return every two months. It immediately occurred to me that the case was one of epilepsy, and I suggested it to the medical man who had sent the patient to me. His answer was that nothing authorised my suspicion, and that convulsions had never been noticed. I still maintained my opinion, however, and shortly afterwards, the poor man had in court a regular epileptic fit, which, unfortunately, left no doubt in anybody's mind; and he was compelled to give up his profession." Although Dr. Trousseau, with his large experience and immense clinical observation, could readily diagnose the epileptic from the apoplectiform state, I feel assured that mistakes may and do arise in reference to this matter; but I have often been led to a right diagnosis by carefully inquiring after the patient's habits and history, particularly in regard to epileptiform conditions, such as sudden and transient attacks of vertigo and unconsciousness; whether there had been nervous twitches preceding the attack, and whether anything like convulsions of the limbs had been observed, or involuntary discharge of urine during sleep; whether the pillow was ever found wet with saliva in the morning, and the tongue sore from having been bitten. I must repeat that it is well to keep these points in view if we would diagnose the condition of the apoplectic or the epileptic with any degree of certainty, and several cases of epilepsy have come under my immediate observation which were said to be cases of apoplexy, but which, by a little careful inquiry, have turned out to be purely epileptic. I remember well the case of a man who was admitted under my care into the Central Sick Asylum, at Highgate, said to have suffered from apoplectic seizures, and on one

occasion I was called to him during the night, and found him quite unconscious, with stertorous breathing ; yet on the following morning he was quite well. A few nights after this he got out of bed, went to the lavatory, seized a poker, and commenced smashing everything within his reach. He was quite unconscious of what he was doing. I had him carefully watched on the following night, and just about the same time he had a confirmed epileptic seizure. I will not here attempt to discuss the question whether, in these cases, the brain is in a state of anæmia, or congestion, it will be sufficient for my purpose if I have shown that cases of apoplexy may be mistaken for apoplectiform cerebral congestion, and I shall, I hope, be able to show you more clearly as I proceed how the one may be diagnosed from the other. A remarkably interesting case of apoplexy from meningeal hæmorrhage came under my care some years ago, which shows how very careful the physician has to be not to jump to a rash or hasty conclusion. I was called hurriedly to the ward to see a young man, aged 22, who was said to be in a fit. I found him lying upon his back quite unconscious, and every muscle of his limbs and face was in a state of clonic convulsion, and although I was somewhat doubtful as to the nature of the attack, I told the nurse to watch him, and then to report to me how he was. I saw the case one hour after this, and the man's condition remained precisely the same, and within thirty-six hours of the time of his seizure this young man was dead. When the calvarium was removed the convolutions of the left hemisphere of the brain were found to be enveloped in blood. In the same ward, and within a few days of the case just narrated, I was called to see a man of about the same age. He was suffering from renal disease and uræmic convulsions, and had I not been aware of this I should possibly have concluded that this man too, had meningeal hæmorrhage. Small aneurismal dilatations of the capillary vessels of the grey matter of the cortex of the brain, as well as of the branches which supply the corpus striatum, are much more frequently the seat of small hæmorrhages which give rise to apoplectiform seizures than, I think, is usually supposed ; and, in making post-mortem examinations in persons over fifty, who have died from some non-cerebral affection, I have observed in connection with these miliary aneurisms the traces of slight extravasations, with degeneration of tissue which must have given rise to

some objective signs during life, although the evidence of these signs might have been of the most transitory character. Some six months since I was travelling in a railway carriage with a well-known public man, over 50 years of age, and whilst we were conversing I noticed some very abnormal movements in the extensor muscles of the thumbs of both hands, and seeing that my attention was drawn to his hands, he asked me what there was to attract my notice, and I then remarked to him in a joking way that his cerebral grey matter was unstable, and that he had abnormal discharging lesions, possibly, from an impaired circulation of the brain. He smiled, saying that he felt a little stupid and faint sometimes, and I was not at all surprised when I heard of this gentleman's death from apoplexy, although, at that time, when I last saw him, he was in the enjoyment of the most robust health.

I will now put the question—Are we justified in coming to the conclusion that certain persons, either from heredity or bodily conformation, are specially predisposed to apoplexy? I should certainly say that such was the case, but with regard to heart disease as a cause of apoplexy, my experience quite bears out the statement of Dr. Walshe, who arrives at the conclusion that hypertrophy of the left ventricle of the heart exerts no agency in producing apoplexy. Generally, hypertrophy of the left ventricle of the heart is associated with mitral or aortic lesions, which are conservative as regards the effects of the augmented power of this ventricle upon the cerebal circulation. There can be little doubt, however, in my mind, that a dilated right heart interferes greatly with the cerebral circulation, and is in a large number of cases, causative, not only of cerebral congestion, but of positive hæmorrhage from the vessels of the brain, and particularly in those cases which occur when the patient is said to be asleep. We know that a dilated right heart is often associated with engorgement of the liver and abdominal circulation ; and no less do we find accompanying this state an engorgement of the vessels of the brain. I believe, however, that such a condition of the heart is equally, if not more, likely to give rise to apoplectiform congestions, miliary aneurisms, and thrombosis, than to hæmorrhage into the brain itself. Fits of apoplexy, we know, do occur after eating too hearty a meal of indigestible food, straining at stool,

violent muscular exercise, sexual intercourse, mental excitement, and so on. Now, in these cases, my impression is that a weak right heart is almost always the predisposing, although not the exciting, cause. Where there is disease in the mitral or aortic valves of the heart, we may then anticipate, should an apopletic fit arise, that embolisms have plugged one or more of the cerebral vessels.

I would venture particularly to impress upon you the importance of never neglecting certain head symptoms, which are premonitory of apoplexy. That these symptoms are due to congestion of the brain I am not prepared to say, although I most firmly believe that they are when they are not associated with an epilepto-genetic idiosyncrasy. What are these symptoms? They are not usually of a severe nature. There is no intense headache, and if the patient goes to his medical man he frequently throws him off his guard by saying that there is not much the matter, and remarking "I only feel a little giddy, and become a little confused in my head at times, and occasionally my head feels heavy, and there is a sense of constriction, as though something were drawn tightly around it, and there is a feeling of stiffness at the back of my neck, and my arms feel heavy, and (a very unusual thing for me) my feet are very cold, and after my meals I feel rather sick, although my appetite is good, and my bowels are acting well. I don't think there is much wrong, Doctor, if you will just give me a little something to act upon my liver." Now, I say that the practitioner of experience will see at once that his patient is suffering from a condition of brain which may at any moment be the seat of such an amount of hæmorrhage as to produce his death in the course of a few hours. Then what course does he adopt to prevent an extravasation of blood on the brain. There are two modes of treatment which, in my experience, should be had recourse to, according to the cause of the symptoms which have just been enumerated, for these symptoms may be produced by arterial congestion with venous deficiency, or they may be produced by venous congestion with arterial deficiency. It not unfrequently happens that the general appearance of the patient will indicate to which of these two states his symptoms are referable. In that state where we find arterial congestion we shall also find increased arterial tension; the pulse will be more or less

full and incompressible, it may be hard and whipcord-like under the finger ; the urine scanty, high-coloured, and loaded with lithates ; the eye may be bright, and the pupils exceedingly active to light. In a case like this I never hesitate to cup my patient freely at the back of the neck, to administer a free mercurial and saline purge, followed by a mixture of ergot and bromide of potassium, perfect rest and a non-nitrogenous diet being made compulsory. On the other hand, where we have a condition of venous congestion with arterial deficiency, we find that the patient will be more or less drowsy and lethargic. The pupil may be contracted or dilated ; it does contract to light, but it does so tardily. The mucous surfaces of the conjunctiva, the lips, the tongue, and the gums will be of a dark venous hue ; the skin will be sweating and clammy ; the pulse will be soft, small, compressible, and variable ; the urine will be secreted plentifully, with a deficiency of urea ; and the heart's action will be exceedingly weak. Now, surely, our treatment in this case will, as a matter of course, vary from that of the condition previously described. I do not consider that local or general bleeding in a case of this kind is at all desirable, and I should not recommend its adoption. The treatment which I usually find most effectual is an alterative mercurial course of blue pill with quinine, to be taken night and morning, and a mixture three or four times during the day consisting of bromide and iodide of potassium, with the tr. of digitalis, and the diet should be highly nutritious and easy of digestion. I hope, in thus placing before you two opposite conditions of the brain, each of which may lead on to apoplexy, both of which, however, differ essentially in their main features, as well as in their treatment, it will not be considered that I have digressed in any way from the main object of my lecture ; and I take the opportunity to state here what I have observed before—"That, if the majority of nervous diseases are to be cured we must study with the utmost care and precision their initial signs and symptoms, and treat these with promptness and courage with the most potent remedies which we have at our command."

I now pass on to the diagnosis of an apoplectic fit. If a man has an extravasation of blood into the medulla oblongata (which is exceedingly rare unless in association with tumour and softening) he falls down and dies instantly. Hæmorrhage into the pons varolii and fourth

ventricle, if extensive, is usually attended with sudden and profound unconsciousness, from which the patient rarely, if ever, rallies. It is well that we should at once bear in mind that there are several other conditions of the brain which may, in many important particulars, be confounded with apoplexy, and to these I shall allude. But previous to this I should like to draw your attention to the main and essential signs which are indicative of the true apoplectic condition. Profound coma is, of all others, the essential feature, and there is, of course, general insensibility to stimuli of all kinds. The cheeks and lips are swollen and livid, or, on the other hand, they may be exceedingly pale, but the face has for the most part a bloated appearance; the skin is more or less cold and clammy rather than hot and dry. The eyelids are closed, conjunctiva injected, and the eyeballs motionless; the pupil will vary according to the seat of hæmorrhage. The state of the pupil is certainly of considerable importance as a diagnostic medium, as, for instance, if there be hæmorrhage into the ventricle of the brain, the pupils will be widely dilated, and no contraction can be induced, either by direct or indirect agency, whereas, if the lesion exists in the pons varolii, the pupils will be found to be minutely contracted; and, in these forms of hæmorrhage, the pupils of the eyes are not influenced by peripheral stimuli, like they are, for instance, in uræmic coma and alcoholic poisoning. If these forms of coma which I have just referred to are not of the most severe type, and of the last degree, peripheral stimulation by the Faradaic brush to the temporal region will produce reflex action of the ciliary muscles. There may be also conjugate deviation of the eyes, to which I shall shortly again refer when speaking of localised apoplexies. The whole of the arterial system appears to have undergone a change; the circulation has become disturbed, and the carotids and temporal arteries will be seen to throb violently. The pulse is usually full and strong, and gives evidence in the majority of cases to the existence of increased tension. The respiration is regular, heaving, or deep, and is for the most part associated with stertor and puffing expirations.

Now these signs and symptoms are significant of a state of profound coma; but it is by no means necessary that these symptoms should be so markedly profound, and just according to the suddenness, extent, and seat of

the lesion, so we shall find the degrees of coma to be more or less marked. The deepest state of coma is produced by hæmorrhage into the pons varolii and into the lateral ventricles. The variations in the body temperature during the apoplectic fit are striking and important. I remember, some years ago, when I had an unlimited field for observation at my disposal, making some experiments in rectal thermometry in these cases, which exactly accorded with the researches of MM. Charcot and Bourneville, at the Salpêtriere, in Paris. The temperatures, pulse and respirations, condition of pupil, and reflex excitability were carefully noted during the three stages —namely, the stage of depression, the stationary stage, and the stage of reaction ; or, as Dr. Bastain puts it—the period of initial lowering, the stationary period, and the ascending period of body heat. The temperature during the stage of depression has been known to fall to 95°F., and during the stage of reaction, or ascending period, it has risen so high as 106° or 108°, whilst the temperature during the stationary period will vary if the patient is to recover from 100° or 103° or even 105° F. We shall find that these variations in temperature, and especially when they are considered in relation to the pulse and respiration, are important guides in our prognosis. If the hæmorrhage is extensive and the depression extreme the patient dies in a state of collapse in the course of an hour or two ; yet, if the patient survives the period of initial lowering he has yet to combat the stage of a too vigorous reaction ; and should the temperature progressively and somewhat rapidly rise to 105° or more, with rapid pulse and respirations, and pronounced coma, there is no chance of the patient's recovery. Then we have the stationary or intermediate period, and we can almost invariably give a hopeful prognosis if, during this period, say, of twenty-four or forty-eight hours, there is merely a temporary rise of temperature, which is soon followed by a fall within the range of the normal temperature of the body. In the coma of uræmia M. Bourneville says the temperature of the body begins to fall at the commencement, and continues to sink as long as the condition persists, so that it may fall as low as 90° F. in fatal cases. On the other hand, in the coma due to cerebral hæmorrhage or softening, the lowering of the temperature is slighter in amount, and in cases not fatal within this period rarely lasts longer than twelve to twenty-four hours. If the hæmorrhage

should occupy one hemisphere of the brain, giving rise to paralysis of the opposite side, we shall find that the temperature of the paralysed side will be from one to two degrees higher than upon the non-paralysed side.

I have referred to conjugated deviation of the eyes. This is a very interesting and pretty constant accompaniment of the apoplectic state. Both the head and the eyes will be, so to speak, driven over towards the non-paralysed side, and in the direction of the brain lesion. M. Provost, who has written an interesting memoir on this subject, states that this condition of conjugated deviation of the eyes and rotation of the head rarely follows lesions of the pons varolii. Both eyes are turned as they would be if the individual were looking upward over one or other shoulder. The eyes are usually motionless, but sometimes they oscillate in the same manner that they are found to do in that peculiar condition of nystagmus which we know to be a constant accompaniment of insular sclerosis of the brain.

Before leavin the question of general diagnosis of the apoplectic state I should like to make one or two observations concerning the differential diagnosis of states of insensibility which simulate a true apoplectic state. Of course there are many causes which produce like effects. In reference to unconsciousness, which we find as the result of hæmorrhage, emboli, and thrombosis of the brain, for instance, we must not forget, when we find a patient insensible, with stertorous breathing, and that this may be due to compression of the brain through an injury. An epileptic falls suddenly in a fit; the effects of the fit pass away, but he may remain apoplectic from compression of the brain caused by fracture of the skull which was due to the fall. It may, in some cases without any history to guide us, be a difficult task to diagnose between opium poisoning and hæmorrhage into the pons varolii. In the former the onset is gradual, whilst in the latter the onset is sudden; we find, however, in each case the minutely-contracted pupils, profound stupor, closed eyelids, cold, clammy, perspiring skin, and complete absence of reflex movements. Uræmic coma will not, I think, as a rule, be confounded with apoplexy; it is usually associated with a pale pasty complexion, puffy eyelids, and œdema of the extremities, and is generally associated with twitching of the limbs, and more or less rigidity, which are not localised; and furthermore, as M. Bourneville has

pointed out, the temperature of the body begins to fall with the onset of uræmic coma, and continually to sink as long as this condition persists, so that it may fall as low as 90° F. in fatal cases. Epileptic coma, which not unfrequently follows the convulsive stage of an epileptic fit, is of short duration ; otherwise it is almost impossible to distinguish it from apoplexy. It occasionally happens that an apoplectiform seizure with convulsions may terminate in a profound apoplectic fit, which sometimes ends in death.

I may remind you that in the majority of these cases the body temperature will aid us greatly in making a diagnosis if we remember that cerebral hæmorrhage is invariably attended in the onset by a marked lowering of temperature, which may gradually rise, even to 108° or 110° F. ; whilst in the apoplectiform seizures which are associated with disseminated sclerosis and general paralysis of the insane the temperature of the patient begins to rise from the first, so that in a few hours it may reach 104° or 105° F., and in fatal cases the temperature rises still higher.

From this and previous remarks which I have made concerning the body temperature in these conditions, I think it will be at once recognised how important it must be in all these cases to satisfy ourselves upon this point before attempting to give a prognosis. It is quite impossible for me, in the course of a single lecture, to discuss and differentiate the symptoms and signs of an apoplectic state due to embolism and thrombosis apart from hæmorrhage, and I assure you that it is not always an easy matter so to do. This much I may say—that thrombosis and embolism are both much more common than is usually supposed, and especially thrombosis. Bastian, in referring to the diagnosis, says—"All that we can say is that a very abrupt onset, in a young person more especially, and in association with the condition named, tells strongly in favour of embolism, and that long or well-marked prodromata, terminating with an attack of hemiplegia in an elderly person points almost as strongly in favour of thrombosis.

Of the different regions of the brain where hæmorrhage may arise we can have no very definite evidence during the apoplectic fit. Should there be central hæmorrhage into the pons varolii the coma is very profound ; the pupils are minutely contracted ; the buccinators are flapped out

with each expiration, and there is an entire absence of sensibility.

Where we have effusion of blood into the ventricles, following hæmorrhage in the neighbourhood of the corpus striatum and optic thalamus the coma is profound, the paralysis general and complete, and the pupils are widely dilated ; tonic and clonic spasms, with rigidity, may exist, and frequently conjugated deviation of the eyes. The anterior lobes are rarely the seats of hæmorrhage; I have, however, met with several such cases. Now hæmorrhage into the frontal lobe of the cerebral hemisphere is not usually sudden, and is not attended with immediate loss of consciousness ; yet it usually terminates fatally if at all extensive. Like other cerebral apoplexies, it is preceded by more or less loss of smell and mental confusion and dulness. Hæmorrhages into the cerebellum are rare, and when they occur are most frequently associated with tumour and softening, so one may say that cerebellar hæmorrhage takes place more often in the young than in the middle-aged or the old. Death is invariable and sudden.

In the treatment of apoplexy it is highly imporant that we should take all the circumstances of the case into consideration. To say that it would be wise or unwise to bleed would depend entirely upon the age and constitution of the patient, the power of the heart, and the force of the circulation. To say that apoplexy is not associated with a more or less congested state of the brain in a majority of cases is to my mind untrue, although I quite believe that many of the cases of apoplectiform congestions are, in fact, no congestions at all, and it matters little to the patients whether they are bled or let alone, for they invariably recover. There is no one who has ever seen a person suffering from the initial stage of apoplexy who has not been struck with the disturbed state of the circulation. If the patient be full-blooded and sthenic with a bounding pulse, which does not yield readily to digital pressure, with the temporal arteries beating violently, with the cheeks red and livid, whether coma were profound or not, I should bleed that patient to the extent of twenty ounces, or I would cup him at the back of the neck to the extent of twelve ounces ; and I should think that I had failed in my duty to my patient if I did not do so. I am not in agreement with those who do not believe that blood-letting prevents or diminishes the cerebral hæmorrhage ; and I

maintain, on the contrary, that where the conditions are such as I have just indicated blood-letting does relieve the force of the heart and circulation, and in this way lessens the arterial tide which has set in towards the cerebral vessels. There is, in cerebral hæmorrhage, vaso-motor nerve paralysis and inefficiency, especially in reference to the vessels of the brain there is increased arterial tension and cardiac force, due to inhibition of the pneumogastric nerve, and by thus relieving the general circulation of blood the vaso-constrictor paralysis which pre-existed is thus removed, and the vessels in a measure regain their normal contractile power and calibre. It is my opinion, gentlemen, that it is quite possible to check a cerebral hæmorrhage by bleeding. I am quite willing to admit that it is useless to bleed or to do anything else if the substance of the pons varolii is destroyed by hæmorrhage, or if the ventricles be distended with blood, but we are not sure, gentlemen, of the extent of either of these conditions until the brain is upon the post-mortem table. During the past seven years I am quite certain, as far as I can well be, that I have saved life in cases of cerebral hæmorrhage by the letting out of blood.

Now, let me draw your attention to the other side of the picture, where I believe blood-letting would aid in destroying rather than saving life, and the condition I would here depict, where blood-letting is contra-indicated is as follows :—A general feebleness and asthenic habit of body, a weak heart, and a soft, weak, and compressible pulse, a cold clammy skin, coldness of the extremities, tips of the nose and ears, and a body temperature below the normal and advanced age. I can see no reason, however, why some patients should not sometimes be bled with advantage because they are old—and by the term old I mean betwec ı the ages of 50 and 65. This is a theme upon which one may dwell for some time, but I must pass on to other modes of treatment. A very good observer, Dr. Austin Flint in his excellent work on "The Practice of Medicine," says that an emetic is sometimes indicated when the stomach is overloaded, and he cites one case where its beneficial influence was well marked. I can only see the object of this reasoning from one point of view, namely, that a quantity of food in the stomach during an attack of cerebral hæmorrhage remains undigested and contributes to collapse, which means vaso-motor paralysis and increased hæmorrhage. I scarcely know of any condition, either of

heart or pulse, when a stimulant cathartic can be otherwise than beneficial, and there is nothing better than two drops of croton oil. It is an old-fashioned but efficient remedy. An injection by the rectum of one pint of gruel, two ounces of common salt, and one ounce of turpentine may be administered with considerable advantage, and without, as far as I can see, any chance of doing harm. There is one special advantage which this injection treatment can lay claim to, and it is this—that it will often clear up a case where the diagnosis is doubtful, and where it is uncertain whether the coma is apoplectiform only or due to drink. We have, again, our asthenic apoplexy to consider where we would not bleed, and where, in fact, I should say wait awhile and do very little, but keep up and maintain the body-heat by wrapping the extremities in flannel, and apply bottles of hot water to the arm-pits and to the feet. If the pulse is very weak and flickering and the breathing slow, injections of brandy must be given, but very cautiously. Injections of ergotine have great power in contracting the vessels, and so arresting hæmorrhage. Dr. Bastian prefers the bromide of camphor to diminish the flow of blood through the brain.

Under any circumstances the patient should be placed in a cool, airy, well-ventilated apartment ; the head should be raised from the small of the back and not jerked up at a right angle with the trunk, as though every effort were being made to close the opening of the glottis ; and if, during the stage of reaction the head becomes very hot cold evaporating lotions and the ice-bag must be kept continuously applied. When the patient merges from the apoplectic state, and when we are led to hope and believe that hæmorrhage has ceased, it is unwise to be too officious ; it would be found that the patient at this time will swallow fluids as automatically as a new-born child, and nothing more need be given than peptonised fluid beef, or peptonised milk. If there be great exhaustion it may be necessary to have recourse to stimulants, and I do not hesitate to say that they may be administered with great advantage in many cases, but unless they are very carefully given I am convinced they may do more harm than good. Let us consider for one moment what is the pathological state of the brain with a foreign body (a clot of blood) in its midst. A brain which is the seat of apoplexy suffers from solution of continuity at the part itself, and also from displacement corresponding to the size of the cavity,

so that of necessity, if the patient is to recover, the clot must be followed by numerous changes, and so also must the brain substance in contiguity with the clot. Under any circumstances the brain substance which is contiguous to the clot must undergo slight inflammatory and sclerosal change, forming a cyst wall ; or, on the other hand, it produces general softening of all the adjacent parts, and I need scarcely say that the treatment of these cases must be directed to prevent this process of softening from extending, so that our great object must be to equalise blood-pressure, to maintain the nutrition of the parts, to improve in every way the condition of the blood, and to secure mental and physical rest.

London : T. DANKS, Steam Printer, 16 Great Windmill Street, W.